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ORIGINAL ARTICLES.

ACUTE AND CHRONIC GLAUCOMA.[1]

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THE treatment of glaucoma, acute and chronic, which is the title of the subject assigned to me by your Committee, necessarily involves some reference to pathologic conditions. It is unfortunate that the term, glaucoma, which is always used in a generic sense, should have been so long retained in our nosology.

I shall assume that acute glaucoma means the occurrence, either primarily or incidentally, in the course of some other affection, sudden increase of tension in the eyeball, with more or less pain, circum-corneal injection, with diffuse opacity of the refracting media, corresponding dimness of sight, with irregular peripheral contraction of the field of vision. These phenomena are usually attended with dilatation of the pupil, profuse lachrymation, sick-headache, photophobia, increased frequency of respiration, pulse, and elevation of body temperature.

[1]Read at the Fourth Annual Meeting of the Western Ophthalmological and Oto-Laryngological Association, held at New Orleans, La., February 10-11, 1899.

Sudden increase of tension of the eyeball, sick-headache and general pyrexia offer two therapeutic indications:

First, prompt and thorough evacuation of the alimentary canal.

Second, active diaphoresis, and diuresis.

Until these conditions have been well established, no sort of local treatment is indicated.

Now, it must not be forgotten that, iritis is frequently a co-existing complication in acute glaucoma, and that it is preceded by mydriasis. In primary forms of iritis, myosis is observed. The local treatment of primary iritis consists in the use of mydriatics; *per contra*, in the local treatment of iritis, incident to an attack of glaucoma, myotics are indicated. Without undertaking to explain the *modus operandi*, it is sufficient to note the fact that, all the myotics are attended by diminished ocular tension, whilst all the mydriatics exhibitit a tendency to increase ocular tension.

It may be noted in this connection that many diverse opinions have been recently anvanced as to the cause of the tension in glaucoma. That which seems to have impressed Dr. Priestley Smith, and in fact a majority of recent writers, is the statement of Treacher Collins, which, though by no means new, is based upon the apparently corroberative evidence that, the so-called spaces of Fontana, in what is now generally called the angle of filtration for the aqueous humor, are occluded by the engorgement of the periphery of the iris, and the coincident contraction of the pectinate ligament upon the posterior elastic layer of the cornea.

Nothing more ridiculously absurd has ever been written in pathology than this baseless hypothesis:

First, the so-called spaces of Fontana are wholly imaginary, being nothing more than the inter-pectineal spaces.

Second, the presence of albuminous matter in the aqueous chambers can not produce increased tension, as may be observed in many forms of traumatism, attended by the escape of blood into the anterior chamber.

Third, the processes of absorption occurring in a normal way, through the lymphatic vessels only, are amply sufficient to remove any albuminous matter from the aqueous chambers.

Fourth, neither the surfaces of the ciliary body, nor the

ciliary processes are able to engage in the processes of secretion.

The aqueous humor is the product of a series of racemose glands found in the projecting part of the ciliary body; it is poured out into the posterior chamber, flowing thence through the pupillary opening into the anterior chamber. A certain equilibrium of pressure is essential to functional activity of all the structures within the globe. If such a condition as the hyper-secretion of aqueous should occur, corresponding hyper-activity of absorption would necessarily follow.

Nothing more ridiculously absurd has ever been suggested than the idea of the phenomena of glaucoma being in any way due to disturbances of either the secreting or osmotic functions of the ciliary body and iris.

It is often observed in the course of mechanical injuries of the ciliary muscles that, increased tension with all the phenomena characteristic of glaucoma, speedily come on. Has it ever occurred to the pathologist that such injuries as bring on œdema, or other effusion of fluid into the ciliary muscle, extending to the ciliary body, or not, must produce at least a perceptible amount of increased tension? Why do we observe peripheral concentration of the field of vision in glaucoma? Is it not clearly due to the extension of œdema or, in other words, the inflammatory effusion from the ciliary body into the contiguous portions of the choroid?

Leaving out all consideration of the traumatic cause, it may be fairly assumed that, the acute attacks of primary glaucoma are not observed in any but the rheumatic or gouty. In all forms of inflammatory rheumatism, the disease begins in the ligamentous or tendinous structures. The ciliary muscle is essentially a ligament binding the ciliary portion of the choroid body to the sclerotica. That it has any other function is mere conjecture. Passing through the ciliary muscle, or ligament, as it was formerly termed, are a series of nerves and small arteries, going to the iris and the projecting parts of the ciliary body. Any disturbance of the circulating blood current in the ciliary muscle is followed quickly by swelling of the muscle and of the ciliary body. This phenomenon is alone responsible for the paralysis of the sphincter pupillæ muscle, which causes it to dilate, for the augmented tension of the eyeball, and for the irregular peripheral contraction of the field of

vision. The increased tension causes the opacity of the refracting media. Pressure upon the nerves passing through the ciliary body gives rise to a sense of pain. The relations of the iris to the cornea can have no possible influence in the production of these phenomena. It is very easily understood anæsthesia of the cornea occurs from the same compression which impairs its transparency. The localized inflammatory process must naturally be inferred from the very beginning of the symptoms. Having produced free catharsis, with profuse diaphoresis and diuresis, the only local treatment which can prove beneficial is section of the iris, which alone will permit free drainage of its engorged and obstructed blood vessels, and indirectly those of both the ciliary muscle and ciliary body.

The experiments of Mackenzie, as early as 1830, demonstrated the futility of paracentesis. If Priestley Smith and Treacher Collins are correct in their assumptions that, excessive aqueous secretion causes the phenomena of glaucoma, evacuation of the aqueous should afford the corrective drainage, and at least diminish or retard the processes of destruction in cases of inflammatory glaucoma. As a matter of fact, evacuation of the aqueous humor has not been found beneficial. Sclerotomy fails because the divided structures soon reunite, and the glaucomatous processes return, usually in an aggravated form, although it may be said in some cases, the subsequent progress of the disease has been retarded. The influence of iridectomy is more likely to be permanent, because the area of divided tissue remains permanently open.

The reason atropin, more than any other mydriatic, provokes attacks of glaucoma in predisposed subjects, may be found in the prolonged effects of the mydriasis upon the coats of the arterioles in the ciliary muscle, keeping up a degree of pressure upon the ciliary nerves sufficient to bring on œdema of the muscle. The reason, therefore, why such myotics as eserine and pilocarpine are beneficial in incipient forms of glaucoma may be found in the prolonged contraction of the arterioles passing through the ciliary muscle, allowing a more complete circulation of the blood current, and tending to prevent that sluggish state of circulation which allows effusion of germs through the walls of the capillary blood tubes. Schlemm's canal is known to be occupied by a plexus of veins, carrying the blood from the iris and contiguous portions of the

ciliary muscle and ciliary body. There are no lymphatic vessels in this sinus. Veins take no part in the work of absorbing fluids, excepting at their points of beginning, just where the capillary tube terminates in the venule, at this point are one, two or three minute valvular openings, which receive fluid of less density that the blood. The processes of absorption occur through the agency of what are called wandering cells, traversing the intravascular spaces, and entering the capillary lymph tubes of all the tissues of the body; these pursue their course to the large lymphatic vessels, and those portions of the lymph stream not used up, in the passage to the glands, are destined eventually to re-enter the blood stream through the right subclavian vein, just as the digested food comes from the intestinal surfaces, through the thoracic duct, into the left subclavian.

It may be fairly assumed that, catalysis must be established as the chief, or the prime factor in the termination of an attack of glaucoma. Defibrinizers and depurants are the agents to be employed; with section of the tissues in confined areas within the limits of restricted circulation.

Chronic Glaucoma.—The phenomena of chronic glaucoma differ from those of the acute variety, in the very mildness and insidious nature of the invasion, and correspondingly in the obscurity from a faintness and uncertainty of the characteristic symptoms. The cupped disc, which speedily appears in suddenly developed and high grades of ocular tension, may be so indistinct in the chronic forms as to escape detection. It is in the chronic stages of glaucoma, which have come on insiduously, that the increased presbyopia appears. It is in this class that those visual disturbances, such as halos surrounding lights, and the imperceptible diminution of the field of vision, goes on until one half, or more, may have been blotted out before the subject is aware of the existence of the disease.

In debilitated subjects, and in neurotic people, chronic incipient glaucoma most frequently occurs. There can be little doubt that the prolonged ciliary irritation of astigmatics and myopes, given to night work, may bring on an insidious or chronic glaucoma. The same may be said of chronic dyspeptics, whose occupation involves prolonged use of the eyes when the nerve centers are disturbed by toxins in the blood. Systematic attention to dietetics, bathing, exercise and the mental

condition demand the first attention in the treatment of such cases. Iridectomy in these cases is rarely, if ever, necessary. Myotics are more important in this class than in the acute forms. The so-called tonic drugs, as iron, strychnine, diffusible stimulants and alcohol are all more or less pernicious, as tending to augment blood pressure through exalted nerve force; whilst some of these agencies retard elimination and are, therefore, more objectionable than others.

It has been found that, in anæmic subjects, both the salicylates and the muriate of pilocarpin may be given in sufficient quantities, and continued to the production of their therapeutic effects, by associating with them fluid foods, as nutritive broths, beef tea, malted milk, etc. If nocturnal headaches accompany the tortuous retinal veins, the dull aching in the eyes, peripheral contraction of the field of vision, even though ocular tension may be absent at the time of examining the patient, the iodide of potassium, in doses of one or two grains, in one half pint of water, every two or three hours during the day, frequently yields brilliant results.

Acute glaucoma is nearly always due to rheumatism, gout, mydriatics or traumatism, and in every instance begins in the ciliary muscle.

The aqueous humor has nothing to do with either the origin or the course of glaucoma. The relations of the iris to the cornea are utterly insignificant, excepting in so far as they may be related through peripheral iritis, incidental to cyclitis.

Increased tension in glaucoma is always the result of inflammatory effusions into the ciliary muscle and contiguous structures, or a slowly developed hyperplasia.

Contraction of the field of vision is probably due to the invasion of the periphery of the choroid by inflammatory effusions extending from contiguous structures.

The tortuous retinal veins and cupped disc are the natural results of augmented pressure upon the nerve structure and the blood vessels against the lamina cribrosa.

The indications for treatment are, such eliminating agents as will reduce blood pressure, and the permanent drainage of the vessels in the constricted areas of contiguous structures by section of the iris. Iridectomy done for this purpose should always be of small extent, including a portion of the sphincter pupillæ muscle, and extending to the periphery of the iris.

The general condition of the patient is of the first importance. Active elimination, alimentary, renal and cutaneous, should be maintained from the start. Eserin or pilocarpin should be instilled into the eyes, provided approximately complete myosis can be secured. Where myosis can not be obtained in half an hour from a single instillation of sulphate of eserin solution, or pilocarpin, in the proportion of one grain to the ounce of water, iridectomy is indicated. Otherwise myotics should constitute the only local treatment. It is hardly necessary to say smoked glasses should be relied upon for protecting the eyes from light.

The patient should, at no time, be permitted to remain long indoors. It is always best to have more or less exercise on foot; this not only promotes digestion and facilitates all the processes of elimination, but it serves at the same time the very useful purpose of diverting the mind of the patient. Easily digested, nutritious foods, without highly stimulating condiments, should be administered regularly and systematically. Copious draughts of pure water should be insisted on, at stated intervals, if necessary. The use of pepper, spices, teas and all alcoholics should be rigidly inhibited. Such drugs as nux vomica, kola, asafœtida, and all cerebro-spinal stimulants should be avoided.

If glandular enlargements, eruptions of the skin or periosteal nodes exist, the iodide of potassium, in doses of two or three grains to a half pint of water, may be administered every hour or two, with beneficial results.

If the tongue is pale and relaxed, and the patient complains of muscular languor, an occasional dose of ten or fifteen grains of the sulphate or salicylate of quinine may be given, with a half pint of beef tea or malted milk. All the glucoses should be forbidden, because they augment the volume of the lymph stream. Electricity, so much praised by some, in chronic glaucoma, has never failed to do mischief, where I have known it tried.

Individual environment must be taken into account as it will often suggest peculiar modifications of the principles of treatment outlined herein.

SAMPLE MISSTATEMENTS IN A BOOK FOR OPTICIANS.

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IT is proper that the physiologist should at times examine and even criticise the books on Physiology intended for public schools and teachers. Likewise it is to be expected that the obstetrician should review the works intended for midwives. It is not in place to state that midwives, like the Indian, are a dying race. The better education the midwife and public receive the sooner the doctor is called and misfortune prevented. It at least will be of some interest, and perhaps service, to the oculists to examine the heresies that may be found in a much advertised book for opticians. It may explain some of the ignorant instruction our patients may have received and assimilated.

OPTICAL TRUTHS. By CHAS. MCCORMICK, M.D,
President of McCormick Optical College, 84 Adams Street, Chicago.

The Keystone reviewed this a short time ago and says the optical truths (?) are stated very clearly. It has been recommended to me by two wholesale opticians as the coming book on optics. As I can not find Dr. McCormick's name in any of our medical directories as a graduate from any reputable medical colleges, I will withhold commend as to his being an M.D. The book has no preface but is dedicated to "the sweet spirit, who has been, is, and ever shall be my *inspiration*." The work commences with an introduction as follows:

"In the practice of ophthalmology one of the essentials is a general knowledge of the nervous system, what it is, whence *it cometh* and whither *it goeth*. In structure it is a series of tubular membranes containing in a minute and continuous *stream*, matter *identical* with the brain substance, through which electrical energy is transmitted. This energy is of two kinds, *galvanic* and *faradic*. The first is generated by the *digestive* organs, and is constant; the second, is an intermittent current generated by molecular friction throughout the body. The nervous system is divided into two classes, the cerebro-spinal or *animal*, and the sympathetic or *organic*."

It needs an active imagination to suppose that the terms

of mechanical electricity may be applied to the body. But this book published in 1898 (near the end of this wide-awake nineteenth century) is largely interwoven with the rankest physiological bosh.

Just here we may look at a sample of the definitions he has given in a separate glossary. In some of these cases there may be seen a slight allusion to *derivations* but absolutely nothing to *definitions*, although this is an age of good *dictionaries*.

"Anæmia—A condition of wasting.

Embolism—A rupture.

Glioma—Glue.

Leucoma—White.

Stroma—Bedding.

Horopter—The field of vision included by both eyes without moving."

The last definition would give an idea that the horopter was the visual field which could be measured with a perimeter. A horopter is the surface of all points seen singly by the two retinæ when the eyes are joined in fixing one point.

He also gives a list of questions and answers to be studied by the reader. Samples:

"Q.—What is light?

"Ans.—It is a force positive and *negative*.

"Q.—Which meridian of the cornea has the sharpest curvature in the normal eye, and why?

"Ans.—The vertical, to give the effect of a plus cylinder to act as a governor to the oblique muscles!

"Q.—Give some symptoms of hyperopia.

"Ans.—Constipation, indigestion, piles, female disorders, almost all nervous disorders, visions more acute than $^{20}/_{XX}$ (!), while myopia has absence of hyperopic symptoms.

"Q.—Why not use prisms for muscle trouble?

"Ans.—Because it is foolish almost to criminality.

"Q.—How much accommodation can one have?

"Ans.—Three dioptres.

"Q.—What is the difference between errors of refraction and muscular insufficiencies?

"Ans.—The first are deformities, and the second is weakness resulting from these deformities."

The author does not explain how emmetropia has these resulting weaknesses.

In referring to refraction he states "there are breaks but no bends." In comment on this I would say, light, in passing through the crystalline lens, does bend, and any study of refraction, to be consistent, ought to be considered from the wave theory and not the old ray theory.

Dr. McCormick advertises: "Our fogging system of testing eyes without atropine is simply a mechanical method of coaxing the ciliary muscle to rest." The term fogging is a very poor one, because a cylinder or a concave lens can cause this so-called 'fog' just as well. Yet several American authors of the irregular type have taken credit to themselves for the name and its application. European and American oculists have long used it. Thus I refer to Morton's (London) little work on "Refraction of the Eye," now in its fifth edition, in which he describes much more clearly and concisely than McCormick, this method. I quote from Morton:

"A method of assisting the patient is to let him put on a pair of + 4 D. glasses for ten or fifteen minutes, and then, without removing, gradually neutralize them by stronger and stronger concave lenses placed in front, until those are found with which the patient can see in the distance the smallest possible type. The manifest hyperopia is apparent by difference in lenses, although atropine is the only safe way to remove all uncertainty."

McCormick speaks of using a + 3 instead of + 4 and this is the only difference. He states this will give the spherical part of correction without atropine. I find he does not mean what he says, for he adds concave cylinders to this to get the astigmatism. Spherical correction has reference to the meridian which is nearest emmetropia. His so-called spherical correction is obtained complete, without the least reference to the astigmatism, which is preposterous. Moreover, he gives advice to stop neutralizing when the vision is almost as good as when the patient can see without glasses. Thus, if the patient could see $^{20}/_{LXXX}$ without a lens he would claim the spherical correction was obtained when $^{20}/_{LXXX}$ or even $^{20}/_{LXL}$ was seen without reference to the astigmatism. Ridiculous!

Instead of stopping at the strongest meridian the patient may stop midway between the two meridians. Thus, with + 2 D. ⌒ — 1 cyl. ax. 45° the patient may stop at 2.50 D. In this case the diffusion image becomes a circle and the lines in

the clock may appear all alike, although the author states dogmatically, if all appear equally distinct the astigmatism is at most very slight. He advises using the stenopæic-slotted disc which is altogether out of date with intelligent American oculists as a final test. Thus, in the case mentioned of + 2 D. ⊃+1 cyl. ax. 45°, if the lens is +2.50 D. which is the spherical lens that probably would be used, the slot would probably stop near 90°, halfway between the meridians, as this section of cornea would be the one nearest correction. The slit hardly ever gives the axis exactly, and as there are at least four great objections to its use, it is surprising that it is still advised in an 1898 text-book.

The above notes may partly explain why so many opticians fall down on astigmatism, especially to be seen in oblique cases. As to spherical correction, they stop before the patient gets full vision, or they buy a machine fixed to overcorrect from one-half to one dioptre, and thus in some cases they overcorrect and in others undercorrect. It is deemed the height of wisdom to add to their correction so as to be able to say they even get more than under atropinizing (McCormick).

As a sample, we will note his advice to be found on page 63: "If — .50 D. ax. 90° gives $^{20}/_{XX}$ vision, prescribe + .50 D. ax. 90° if it gives $^{20}/_{XXX}$ vision, even if the first was found under a mydriatic." Here is a man, president of an optical college, and supposed to have some optical education, who actually proposes in a case of astigmatism, *against the rule*, to correct it as if it were astigmatism *with the rule*.

In regard to muscle troubles he makes such puerile statements as the following: "The eyes in muscular insufficiencies always deviate the same way the lights do."

"If the *base* of the prism is towards the nose it proves the internal recti are too active. The weak muscles are always under the apex of the prism."

He states that he is aware that in regard to the tests for exophoria, he stands alone, "but all the evidence supports my position."

He draws a curious diagram dividing the lens into two prisms and selects the portion to suit his whim, and forces the ray through that.

He admits that in actual cross-eyes facts oppose him, but says there is a limit angle before coming to that. The *cover-*

test ought to show any reasonable man how the eyes turn.

He states, glasses have often wonderful cosmetic effects, removing wrinkles, etc. "The contraction of the sphincter muscles throughout the body in hyperopia causes menstrual difficulties in females, and piles in both males and females. Glasses will remove these" (!). "If a patient has $^{20}/_{xx}$ vision he has no disease which affects the eye."

Any oculist finds serious diseases of the eye often with as good vision as that. I am now treating a patient whose fundus is spotted like a checker-board from an old syphilitic chorioiditis whose vision is often better than $^{20}/_{xx}$ but with relapses.

"After the eye has matured, at 10 years of age, if the correction is equal to the error *no* changes of lenses will ever *be needed.*" A great mistake, as even the batting of the lids may change the shape of the eye in a few years.

He devotes a chapter to "Exposing Ophthalmological Charlatans and Their Practices," and takes as his motto: "There are tricks in all trades but ours." He refers to the "indirect method" as though it was a humbug, by which, at best, only an inverted image can be seen. The microscope and telescope use an inverted image and yet they are used!

"The cause of most cases of conjunctivitis is hyperopia, which causes the sphincter nerves and muscles at the mouth of the Meibomian gland to contract, and the matter is forced back between the conjunctiva and lids which become *granulated.*" McCormick could probably not explain how granulated lids are almost unknown in some places and among some races. Yet he rants about the gross criminality and ignorance of those who use escharotics on granulated lids. "It is a crime against Nature."

On page 85 he states: "There is a publication which advertises itself as the greatest of its kind, and says 'its editors and contributors are the cream of the profession.'" "On page 5 of its January, 1897, issue, the *Ophthalmic Record* has a contribution under the heading 'Metamorphopsia,' in which seven cases, measured under atropine, are recorded and the lenses prescribed are given." There were also cases of anisometropia as described by a Tennesse writer. He, of course, did not give full correction but merely gave what the patient accepted. McCormick spends two and a half pages and a dozen diagrams to prove that the eyes were not corrected! He then adds: "If

such are the cream of the profession, for humanity's sake, let us have skimmed milk."

He mentions a prescription "from one of the experts at the Illinois Eye and Ear Infirmary. It was written on one of the cards of admission to the poor: R. V.=$^{20}/_{XXX}$, L. E.=$^{20}/_{L}$, and the two meridians were shown on a cross. It was sent to me by an optician for interpretation. I presumed the patient was the victim of a retinoscope fiend and that the lines meant the meridian of the eye. But I suggested that as vision was so much worse with the right, it was evident the prescription was not an approximation." A queer conclusion that one eye could not be $^{20}/_{XXX}$ and the other $^{20}/_{L}$. This was not a prescription at all but probably a memorandum of part of a test which the patient had got hold of. The Infirmary had better trade with some optical house that can telephone for full directions rather than have to depend on such an adviser.

He devotes a chapter to "Operations, Medicines, and Prisms—Three Great Blunders." He has no use for these, as he thinks headache, insanity, dyspepsia, piles, nervous prostration, and female diseases can be cured by correcting with a convex glass the hyperopia.

Just here he gives another example of his fantastic mathematics: "A hyperope of 2 D., in order to focus light from infinity upon the retina, must accommodate 2 D., and the normal tendency to converge to the twenty-inch point, with that much accommodation, would cause diplopia but for the demands of the 'guiding sensation' which calls for sufficient power to be sent to the external recti through the sixth nerve to prevent that convergence. This requires, at least, 2 D. of nerve force, thus we have 2 D. accommodation in each eye, 2 D. convergence and at least 2 D. to restrain that convergence, making a total of 8 D. per second. So figuring, as in the other example, we have $8 \times 60 = 480$ D. per minute; $480 \times 60 =$ 28,800 D. per hour; and, as the hyperope uses his accommodation all the time, for sixteen hours daily, we have $28,800 \times 16 = 460,800$ D. per day. Thus, if he has the same work to do as the emmetropic, we add the 97,200 D. to the 460,800 D. making a total strain upon the nervous system of the hyperope of 558,000 D. daily. Compare this with the normal 97,200 D. The extra 460,800 D. must come from the reserve supply stored in the brain, and when that is exhausted trouble begins."

Shades of the Keeley motor! If some soldier could have extracted some spare 558,000 D. from the brain of some optical pseudo-professor it might have helped in demolishing the whole Spanish fleet. His mathematics are more whimsical than the opposite idea which says a 2 D. of hyperopia represents an eye too short by only one-half a millimeter or less than one-fiftieth part of an inch and therefore of not so vast an importance.

Here is another outburst of wisdom: "I will make the broad assertion that as long as sufficient vitality remains to keep the afflicted out of bed, even in chronic diseases, the correction of the hyperopia will effect a complete cure in 90 per cent. of all cases." As to hyperopia, he asks, "if such unnatural effects would not be in proportion to the amount of error." He says, emphatically, "Yes." In this McCormick is much astray. Patients with a + 8 D. hyperopia may not have any eyestrain or headache which may be so prominent in cases with + 1 D.

In his chapter on "Diseases of the Eye," he states that "keratitis and iritis are due to errors of refraction." He says, "in glaucoma the disc is of greenish hue, hence the name glaucoma (green)." The word glaucoma has no reference to the optic disc but merely to the reflex which may be seen in many conditions with a dilated pupil and slightly dimmed media. It is a very mischievous doctrine for McCormick to give the impression that the disc or anything else is always green in glaucoma.

He speaks of inflammation of the retina being due to sympathy. "This is called retinitis. It is not a disease, but a *symptom!*"

In reference to the ophthalmoscope he states: "The operator should first familiarize himself with the appearance of the *normal* retina (see cut)." He gives two nearly full-page plates, both alike, except one is cheaply colored. On both there are some thirteen *retinal* vessels which do not reach the optic disc but spring up from all parts of the retina. Whoever made these plates never saw a human retina, and I know of no animal that has such odd retinæ as here figured. The coloring on a tomato-can would excell in faithfulness.

He says the "absence of the light streak in the veins is due to the opacity of the blood which absorbs the light." It

further strengthens me in the idea that the author never saw a human retina, as he would have seen the light streak on veins during his earliest examinations.

In speaking of "thread-like vessels, pale arterial blood, and dark venous with general appearance of lack of proper nourishment," he says, "it indicates disorders of the generative organs in females and functional derangement of the liver and digestive apparatus generally in males." Some men are visionary on the subject of female organs. Surely what would affect the liver and stomach in man would affect the same in woman. He states "the *commonest* cause of female ills is hyperopia and ignorance of their functions."

On page 112 he states: "In hyperopia the constant accommodative effort causes a sympathetic contraction of the sphincter muscles throughout the body (and it must be remembered every opening in the body, large or small, is surrounded by such a muscle), among them that of the uterus, thus interfering with the menstrual function. This sets up a disturbance which is reflected back to the eye, causing further contraction of the muscles there, even to those surrounding the optic nerve at its entrance, with the result that the arterial circulation is cut off so blood can not enter the eye, venous circulation is impeded so blood can not pass out, and the field around the edge of the disk becomes so irritated that the disk appears like a splash of cheap whitewash, tinted. The cut was drawn from such a case." In comment, it is in place to say there is no sphincter-muscle surrounding the optic nerve papilla. A full-page colored plate at the beginning of the book represents a cartilaginous cushion closely hugging the optic nerve in immediate connection with the eye. This cushion is attached to nothing except the recti muscles which are extremely short, not half the circumference of the eye. It would be a surprise to the Doctor to know that the optic foramen is an inch behind the eye and that there is no sphincter cushion surrounding the nerve at the papilla.

The cut which is supposed to represent the effects of hyperopia is really that of a serious disease, *choked disc*. The striation of the nerve is enormously increased and the margin of the disc can not be made out. This is fine teaching for students! Many an eye in such cases would be forever lost through a few weeks' delay with glasses and ignorant pre-

sumption. Sad to relate, every oculist has the histories of just such cases, who have lost their sight irretrievably while wasting time trying several changes of glasses.

In speaking of patches in the eye, he says "if these appear black, as they will when the degenerated tissue disappears, showing the pigment layer, it is safe to predict death. If your prediction fails, the patient is pleased, and if it is fullfilled, your reputation is enhanced." Comment: It is safe to doubt, the patient will be pleased when he may find from any oculist that pigment spots both in retina and choroid, have nothing to do with prognosis as to death.

McCormick states there are no nerves in the sclera. Königingstein, as early as 1881, gave an accurate description of the scleral nerves. Dr. McCormick also describes "the zone of Zinn as a tubular structure." This is an exploded fallacy!

In his chapter on "Color-Blindness," he tries to prove with a diagram that color-blindness is due to the fact that the lens is not achromatic. He states that it is impossible to find a color-blind person with $^{20}/_{xx}$ vision. I have found them with $^{20}/_{x}$ vision and so have other observers. A lens that is not achromatic would not change monochromatic light. Thus, a red-blind person in a red room lighted by a red light would not perceive the redness, although a lens not achromatic could have no such effect in causing the trouble. He states that the only modern method of testing color-blindness is a method devised by one of his assistants; Prof. Rumble's method by rotatory circles is a very poor and imperfect imitation of Jeaffrieson's color circle, an English invention.

Dr. McCormick has a statement on the last page of the book that his college gives the most thorough course of optics in the world. Commencement, $40, graduation and diploma all in a week.

I have given but a few of the many heresies which the book contains, but which an innocent public may be supposed to assimulate.

A CASE OF ATROPHY OF THE OPTIC NERVES FOLLOWING HÆMORRHAGE FROM THE STOMACH, WITH A CONSIDERATION OF THE CAUSES OF POST-HÆMORRHAGIC BLINDNESS.

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LOSS of vision as a consequence of hæmorrhage from the stomach is an occurrence of sufficient rarity to warrant the reporting of every well-authenticated case of this character. In a paper on "Amaurosis and Amblyopia after Hæmatemesis," by Dr. Ed. Pergens, of Brussels, in the January, 1896, number of the *Annales D'Oculistique*, the author, after a seemingly exhaustive search of the literature of the subject, has been able to bring together data of but sixty-four cases, two of these being newly-reported cases of his own.

The unanimity with which the text-books upon diseases of the eye mention excessive hæmorrhage, and especially hæmorrhage from the stomach, as one of the causes of optic neuritis, would seem to indicate that cases of this character are not of infrequent occurrence—a conclusion scarcely warranted, I am inclined to believe, by the facts.[1]

Briefly reported, the case which has recently come under my observation, is as follows:

A. B., 57 years of age, formerly a lumberman of West Virginia, and more recently a restaurant-keeper in one of the small towns of that State, was seen December 5, 1898. He gave a history of serious disturbance of the stomach of twenty years' duration, the most prominent symptom being frequently repeated attacks of vomiting. On May 6 (1898) he had, for the first time, a hæmorrhage from the stomach. It was of a severe character, and was followed, at intervals of forty-eight

[1] I have been able to find in the Catalogue of the Surgeon-General's Library, under the title "Inflammation of the Optic Nerve," but a single reference to optic neuritis following hæmorrhage.

hours, by two other equally severe hæmorrhages. The loss of blood was so great that his life was despaired of, and he was confined to bed for six weeks. For part of this time (two or three weeks), according to his account, he was in a semi-conscious state—was aware of the presence of people about him and could hear their voices, but could not speak to them or express his wants.

On the day of the second hæmorrhage, his sight, which had previously been good, became greatly impaired, and his belief is that during the two succeeding weeks he was "entirely blind." At the end of this period his vision began to improve, and he was able to see people moving about the room. This improvement in vision continued, so that by the last of June or first of July he could see well enough to walk upon the streets without guidance; and this amount of vision he retained until about the middle of November, when his sight began again to decline. The decline from this date was pretty rapid, so that when I saw him he had to be led about like one entirely blind.

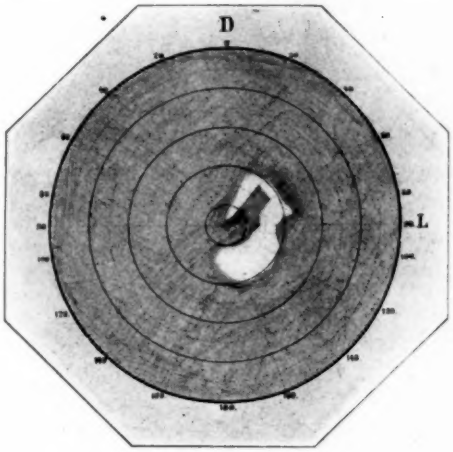
He admitted that he had been a pretty constant smoker, but denied having been a hard drinker, and also denied having had syphilis.

The condition of his stomach was carefully investigated by Dr. Osler and Dr. Thayer, who found a nodular mass near, and partially occluding, the pyloric orifice, which they regarded as the result of a chronic ulcerative process, the indications pointing to a more recent development of a malignant growth in the old cicatricial tissue.

The examination of the eyes gave results as follows: Pupils semi-dilated, somewhat oval in shape and entirely unresponsive to light. The ophthalmoscope showed advanced atrophy of both optic nerves, with some cupping, and marked contraction of the retinal arteries. The optic discs had a woolly appearance, their outlines were irregular and ill-defined, and in each eye there were pigment changes in the retina, not only about the margin of the disc but at points some distance from from it and especially in the macular region, indicating that the atrophy of the nerves had been preceded by an inflammatory process which had involved the retinæ as well as the optic nerves. Upon testing his vision, I found that with each eye he was able to count fingers at about 12″, but only in a very lim-

ited part of the field, which in each eye was slightly to the temporal side of the central fixation point.

After an interval of eight days, he having meantime been under treatment in the Johns Hopkins Hospital, he thought his sight somewhat better, and I found that he could then distinguish with each eye Snellen C. at about 10″. An attempt was made at this time to take his visual fields; but this was very difficult and the result unsatisfactory because of his macular blindness and consequent inability to maintain central fixation. The result obtained in the right eye is shown in the accompanying diagram; the attempt as to the left eye was



abandoned Although there seemed but little probability that his sight could be improved by any plan of treatment, he was placed upon small doses of hydrarg. biniodid. with potassium iodid., and increasing doses of strychniæ sulphas.

Although, as has been said, the text books, almost without exception, speak of loss of sight following severe hæmorrhage from the stomach, usually ascribing this result to optic neuritis, they have but little to say as to the way in which the loss of blood induces such disturbances in the visual apparatus.

The theory, advocated by Samelsohn[1] and others, that the optic neuritis is not due to the loss of blood, but that it and the diseased condition of the stomach which induces the hæmatemesis are both dependent upon a central lesion, probably

[1]Graefe's Archives, Vol. XXI, No. 1, p. 150.

in the optic thalamus, does not seem to be tenable; for we know that other severe hæmorrhages as well as those from the stomach are followed by loss of sight. Thus Fries[3] states that while 35.5 per cent. of the reported cases of amblyopia from loss of blood were due to hæmorrhage from the stomach and intestines, 25 per cent. were due to uterine hæmorrhages, 25 per cent. to abstraction of blood, 7.3 per cent. to epistaxis, 5.2 per cent. to bleeding of wounds, and 1 per cent. each to hæmoptysis and hæmorrhage from the urethra.

The theory of Westhoff and Ziegler, that the loss of vision is caused by a primary fatty degeneration of the optic nerve induced by ischæmia, as well as that of Hoffman, who attributes the amblyopia and the subsequent atrophy of the optic nerve to a retro-bulbar neuritis, seem to have received but little support from the evidence afforded by the ophthalmoscope in the majority of the reported cases.

In the paper of Pergens, already referred to, a brief abstract is given of each one of the sixty-four cases of amaurosis and amblyopia following hæmatemesis which he was able to find upon record. In a considerable number of them no ophthalmoscopic examination was made; in forty-three instances the ophthalmoscopic findings are given, but the time at which the examination was made varies greatly in different cases.

If we decide, arbitrarily, to regard all the ophthalmoscopic examinations made within three weeks of the onset of the eye symptoms as *early*, and all after this period as *late* examinations, it will be found that twenty of the forty-three cases belong in the first category and twenty-three in the second. The early examinations are, of course, the more instructive.

Now, after a careful consideration of the findings in these earlier examinations, it seems to me that, while a very fewof them might, perhaps, be cited as supporting the theory of primary fatty degeneration of the optic nerve of Westhoff and Ziegler, the great majority of them point strongly to an obstruction of the blood current in the central retinal artery as the cause of the subsequent intra-ocular manifestations; and, in view of the well-recognized tendency to the formation of thrombi in post-hæmorrhagic anæmia, it seems highly probable, if this theory be correct, that the obstruction was of thrombotic origin. This

[3] Klinische Monatsblätter für Augenheilkunde, 1878.

seems the more probable, because there is, I believe, a reason why the disposition to thrombosis after loss of blood should manifest itself especially in the retinal vessels.

The occurrence of thrombi after excessive hæmorrhage is to be explained by the reduction of blood pressure and the consequent slowing of the blood current, the alteration in the condition of the blood itself (especially the multiplication of the platelets) and, probably, also by changes (consequent upon anæmia) in the vessel walls. Now, in the retinal circulation, beside all these general conditions, we have, in the intra-ocular tension, a special condition tending further to obstruct the enfeebled blood current. Here then, it would seem, at the point where the central retinal artery pierces the lamina cribrosa and becomes subject to the intra-ocular pressure, the ideal conditions for the development of a thrombus exist; for here we have the especial point of constriction or obstruction behind which, when other conditions are favorable, a thrombus is prone to develop. The intra-ocular pressure, doubtless, impedes the blood stream in the retinal veins also, but, probably, not to the same degree as in the arteries.

The ophthalmoscopic picture in thrombosis of the central retinal artery is much the same as is found in embolism of the artery, namely, paleness of the optic disc, marked contraction of the retinal arteries, less marked contraction of the veins, opacity of the retina, especially about the disc and the macula, a cherry-colored spot at the macula, and, occasionally, hæmorrhages; indeed, there are grounds for believing that many of the cases which in the past have been regarded as *embolism* of the central artery of the retina were, in fact, cases of *thrombosis*.[4]

Having in mind this picture, let us consider, more in detail, the ophthalmoscopic findings described in Pergens' paper and see in how far the two agree. In the twenty earlier examinations, which, as before stated, are the more instructive, we find recorded the following conditions:

	Cases
Arteries contracted	5
Vessels contracted, especially arteries	2
Vessels contracted	2
Arteries filiform, veins very thin	1

[4] Kern, Zur Embolie d. Art. centr. Retinæ. Inaug. Diss, Zürich, 1892.

Arteries contracted, veins dilated	3
Total in which there is mention of contracted arteries	13
Optic disc pale	11
Optic disc greenish-grey	1
Optic disc clouded	2
Optic disc clouded and white	1
Total in which there was ischæmia of the disc	15
Cloudiness of the retina	7
Cloudiness of the retina with cherry-colored macula	2
White plaques, miliary exudates, etc., in retina	3
Total in which there were characteristic changes in the retina	12
Hæmorrhages in retina or upon optic disc	8

Here we have, in at least three instances, the typical picture of obstructed arterial circulation—the case in which the arteries were filiform, and the two cases in which there was clouding of the retina with the red spot at the macula—and in all of the others it is safe to say, I think, that the conditions present may be accounted for more satisfactorily upon this theory than upon any other.

Besides these there are two cases in which the retinal vessels are described as normal (vision being recovered in each); one in which the arteries were slightly enlarged and tortuous and the veins greatly so; one in which the veins were dilated and hæmorrhages were present in each eye; and one in which there was a picture of "arrested circulation," all the retinal vessels being increased in size. In the three last-mentioned cases the indications point to interference with the venous circulation rather than with the arterial. This might be explained by the occurrence of a hæmorrhage into the sheath of the optic nerve, as suggested by Samelsohn, or by thrombosis of the central retinal vein, although it can not be said that the characteristic signs of this latter condition were present in any of these cases. In this connection, however, it is of interest to mention a case of marked impairment of vision following a severe uterine hæmorrhage, observed by Dr. Harry Friedenwald, of Baltimore, in which the ophthalmoscope showed in

each eye the typical picture of thrombosis of the central retinal vein. The vision of one eye was regained, but that of the other was permanently lost.

As to the evidence afforded by the ophthalmoscope in the twenty-three cases described by Pergens, in which only a late examination was made, it can not be claimed that it throws much light upon the question under consideration; for the atrophied nerves and contracted arteries usually mentioned as present might have been due to other conditions as well as to thrombosis of the central artery, and this is equally true of the case which I have reported.

The character of the visual field in my case, as well as that in several of the small number of cases in Pergens' paper in which the field is described (vision having been retained only in a circumscribed area in the temporal field) is significant, and seems to point to the partial preservation of the retina in the neighborhood of the papilla by means of the cilio-retinal arteries.

Of the whole number of cases collected by Pergens, autopsies were made in but four. In one of these a thrombosis of the splenic artery was found, but no mention is made of the condition of the eye. In a case reported by Hirschberg[5] there was complete atrophy of the optic nerve of one eye, and atrophy of a limited portion of the optic nerve of the other eye. In the affected portion of the nerve there were numerous blood vessels with thickened walls, but no thrombosis and no signs of hemorrhage in the optic nerve sheath. The death of the individual, it should be stated, did not occur until three years after the loss of vision.

In an autopsy by Ziegler,[6] twenty days after the attack which led to loss of vision, no macroscopic changes in the optic nerves or their sheaths were found; but the microscope showed fatty degeneration of the nerves and their intra-ocular expansion.

The only other autopsy was one made by Raehlmann.[7] All the arteries presented constricted lumens from a fibrous endarteritis. The veins also had undergone slight constriction,

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[5]Zeitschr. f. Klin. Med., Vol. IV.

[6]Ziegler und Nauwerck's Beitr. z. path. Anat., Vol. II.

[7]Fortschr. d. Mediz. 1889, p. 928.

in two places being almost totally obliterated. There was œdema of the retina, especially in the neighborhood of the disc. In the choroid the endarteritis was pronounced and there was hyaline degeneration.

Here, too, it will be seen, we have mention of vascular changes, the thrombosis of the splenic artery in the first-mentioned case being, at least, suggestive, and the condition of the retinal vessels and the retina itself in Raehlmann's case being especially significant.

The fact that both eyes are so frequently involved in blindness depending upon acute anæmia[8] seems, at first sight, to work against the theory that the loss of vision is due to thrombosis of the central retinal artery, since it implies the occurrence nearly simultaneously, at different points, of two thrombi; but, if the intraocular tension plays as important a rôle in the etiology of these cases as I believe it does, this objection loses much of its force.

Conclusions.

That the weight of evidence afforded by the ophthalmoscope points to thrombosis of the central retinal artery as the usual cause of the blindness which occurs in post-hæmorrhagic anæmia.

That the resistance offered to the already enfeebled blood current in the central retinal artery by the intraocular tension is an important etiological factor in determining this result.

That, in exceptional instances, the ophthalmoscope indicates that the thrombosis occurs not in the artery but in the central retinal vein.

That, in other exceptional instances, it may be that the loss of sight and the ophthalmoscopic changes which accompany it are the result of a hæmorrhagic or serous effusion into the optic nerve or its sheath (Samelsohn). And here, again, the obstruction and damming back of the blood current in the central retinal artery by the intra-ocular tension, probably, have much to do with bringing about this result.

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[8] Whether one or both eyes were affected is stated in fifty-seven of the cases collected by Pergens. Of these both eyes were involved forty-nine times; one eye only eight times.

A CASE OF ATYPICAL RETINITIS PIGMENTOSA[1].

BY HOWARD F. HANSELL, M.D., PHILADELPHIA, PENN.

THE report of the case briefly described below was suggested by the article by Fuchs in the *Archives of Ophthalmology*, September, 1898, and is offered as a small contribution to the subject of hereditary atrophy of the optic nerve, retina and choroid. Until the appearance of Cutler's paper, *Ibid.*, July, 1895, in which four cases of hereditary night blindness studied in Fuchs' clinic were reported; all cases of this kind were classified under the general title of retinitis pigmentosa, but Fuchs has shown that this disease is only one form of the affection. He has described retinitis albescens, circular atrophy of the choroid, retina and degeneration of the optic nerve, and others have reported cases of retinitis pigmentosa without pigment. All have practically identical clinical histories and symptoms but differ in their ophthalmoscopic appearances. They are characterized by night blindness—the earliest sympton, by progressive atrophy of the retina, absorption of the retinal and choroidal pigment, and wasting of the optic nerve. This classification and a further elaboration of it seems to me a desirable advance in our studies of diseases of the eyeground in young subjects. We have been accustomed to name the diseases according to their ophthalmoscopic findings only, and have omitted the important consideration of their histories and pathology. Thus, a single large patch of choroidal and retinal atrophy is called "choroiditis areolaris," numerous isolated patches "choroiditis disseminata," etc. These expressions convey an idea of the ophthalmoscopic findings only, and disregarded the underlying causes, family and personal history, complications, course and final outcome.

Wagenmann, *Graefe's Arch.*, Bd. XXXVI and XXXVII, states that in pigment degeneration of the retina the initial disease is choroidal, and since the retina, or at least its posterior layers depend upon the choroid for its nourishment, choroiditis disseminated throughout the entire fundus would indicate

[1]Read before the Section of Ophthalmology, College of Physicians, Philadelphia, March 2, 1899.

gradual and general atrophy of the posterior layers of the retina and slow loss of function. The difference between the various forms of choroiditis and the modified forms of congenital and probably inherited retinitis pigmentosa, described by Fuchs is, that, to the atrophic process commencing early in life in the choroid, is added slow and generally progressive optic nerve and retinal atrophy, giving rise to the early night blindness and the later complete loss of function.

The characteristic features of the three forms of modified retinitis pigmentosa, condensed from the papers of Cutler and Fuchs, are as follows:

Retinitis punctata albescens. White points sharply limited and without pigment, most numerous around the papilla and macula, disseminated throughout the retina *in connection with* existence of the disease in childhood, blood-relationship of the parents, diminution of direct vision and marked concentric narrowing of the fields and night blindness—the last, since childhood.

Atrophia gyrata choroideæ et retinæ. Atrophy of the papilla, retinal vessels almost invisible, papilla surrounded by a zone of normally colored fundus, this by an atrophic area containing islands of normal appearance, joined to each other by long narrow bridges—the remains of healthy fundus left between the atrophic areas, pigment spots toward the periphery, night blindness, consanguinity of parents. The choroidal atrophy is the most conspicuous feature of the ophthalmoscopic picture. "This occurs first discreetly in the form of round, sharply margined bright dots which increase in size until they become confluent. They now melt completely into one another or remain divided by small strips of normal fundus. Over the atrophic area a few choroidal vessels and pigment specks are to be seen. The atrophic spots lie in a zone having the papilla for the center and extending from the equater to this point. The longer the disease lasts the nearer this zone approaches the papilla." Posterior star-shaped cortical opacity of the lens was found in all of Fuchs' cases.

Retinitis pigmentosa without pigment The symptoms and history of the cases reported, of which there is a sufficiently large number, do not differ from those of the typical pigmented forms. Its characteristic features are consanguinity of parents, early and marked night blindness and progressive

atrophy of the optic nerve and retina. The bibliography, from the earliest ophthalmoscopic times, is given by Cutler. Since the publication of his article, papers, descriptive of the disease, have been written by Simonds (*British Medical Journal*, January 22, 1898); Gould (*Annals of Ophthalmology*, October, 1897); Atwood ("Royal London Ophthalmic Hospital Report," 1895), and others.

CASE.—Anna H., aged 10 years; a pronouced blond, but healthy-looking child, of healthy parents who are not blood-related. H. 5 D. The media are clear, the lens showing no signs of cataract. In the right eye, between the disk and papilla is an oblong pigment patch, two-thirds the size of the disk, surrounded by a white ring of atrophied choroid and nearer the periphery are several smaller round spots of partly atrophied choroid. The optic nerve is distinctly paler than normal, the veins of good size but the arteries contracted. The field is slightly limited toward the temporal side. The left eye shows the same general appearance, although the pigment patches are fewer and less noticeable. V. $^{20}/_{50}$. The child has not yet complained of night blindness, partly because of her youth and partly because the disease is in its incipiency.

I classify this case among the anomalies of retinitis pigmentosa mainly because of the atrophy of the optic nerves commencing in childood and existing in connection with unusual patches of atrophied choroid and retina found in both eyes. This diagnosis may be open to question from the feeble characteristics of retinitis pigmentosa, yet I believe at a later stage of its development the diagnosis will be confirmed.

AN OCULIST'S EXPERIENCE IN THE ARMY.[1]

BY JOHN KYLE, M.D., MARION, IND.,

MAJOR AND SURGEON 160TH INDIANA VOLUNTEER INFANTRY.

THE emergency which demanded the calling to arms of some 160,000 volunteer soldiers, and the rapidity with which they were mobilized, astonished the world. Considering the rapidity with which regiments were recruited, the physical, intellectual and moral standard of the troops in general was such as to thrill the hearts of those who had the opportunity of being closely in touch with our army. It will be a growing source of pride, as in after-years, history relates the true, unbounded and unselfish patriotism of the American people.

To begin with, I want to disclaim that the position of an oculist, suddenly transported to the full charge of a regiment, brigade or division, is an enviable one. To me the transition was very sudden; to step down from an easy-going business to all the laborious duties of an army surgeon in the field, with books, records and reports to learn and supervise was quite enough to turn beautiful black hair to silver whiteness. However, medicine and surgery when once learned are never quite forgotten, while records are easily learned.

The rapidity of mustering the respective regiments into the United States service and the demand that all be taken, gave but little time for a thorough and complete examination of the eyes. But few surgeons were at all familiar with this organ; the consequence was that many men entered the service with greater or less eye affection. Among the more prominent defects which I have observed were convergent squint, double and single coloboma of the iris, mydriasis, retinitis pigmentosa, myopia, hyperopia and dacryocystitis. The question will naturally arise, why men with defective vision could slip by the examiner without being detected? The majority of young men who presented themselves were quite smooth and quick to impart information to any of their unfortunate

[1]Prepared for the Fourth Annual Meeting of the Western Ophthalmological and Oto-Laryngological Association, held at New Orleans, La., February 10–11, 1899.

companions. I especially remember the cadets from the University of Vincennes, all madly enthusiastic, when one of their members with defective eyesight was stood up for examination, one or more constituted themselves a committee to whisper information through the cracks of the wall. Every scheme was resorted to to escape the vigilance of the examining surgeon.

In making the examination of a recruit the color of the eyes is determined by comparison with the standard eye-colors issued by the Surgeon-General. Note is made if the color is not the same in both eyes, and if there are any peculiarities of pigmentation, that is, the presence of black or red spots in the general color, and if there is any deviation in the normal circular form of the pupils.

The Medical Department of the Army has classified the different diseases, and in consequence the surgeon is in many cases restricted in his nomenclature. This does not, however, apply so much to the diseases of the eye. I will enumerate the diseases as recognized:

Diseases of the lids, acute and chronic conjunctivitis, corneitis, diseases of the choroid, iritis, retinitis, snow blindness, asthenopia, cataract, glaucoma, amaurosis and night blindness.

It will thus be seen that the many and varied forms of eye disease are not expected. In consequence the regiments are poorly equipped with instruments necessary for delicate operations upon the eye. However, all general hospitals are amply provided with such instruments. I do not appreciate the reason why the surgical outfits supplied to the regiments for field service are not furnished with a Politzer bag. This is one of all the instruments every surgeon with the army in the field has a demand for daily. The diseases which I have most frequently observed, are the following enumerated in order of frequency:

Acute and chronic conjunctivitis, keratitis, pterygium, hyperesthesia of retina, granular conjunctivitis, optic neuritis and acute glaucoma.

It has been a source of interest to me that so few cases of iritis, keratitis and granular conjunctivitis have come under observation during the past year. When we take into consideration the climatic changes, the great amount of dust troops on the march are exposed to, the scarcity of cases of conjunctivi-

tis is wonderful. In the field it is impossible to make a microscopical examination of the cases of conjunctivitis. Unfortunately microscopes did not reach us until very late at Chickamauga Park, and I thus had not time to devote to the work. Considering the fact that gonorrhœa is such a prevalent disease in the army I have so far seen but five cases of gonorrhæal conjunctivitis. In Matanzas, Cuba, a great many cases of acute conjunctivitis presented themselves for treatment.

Dr. Gifford, of Omaha, has, in a number of interesting articles, called especial attention to the pneumococcus of Fraenkel as a cause of a majority of cases of conjunctivitis in that city. Since reaching Cuba I have carefully examined, microscopically, every case of conjunctivitis in my regiment for this organism to, if possible, prove that the pneumococcus is the most frequent cause of conjunctivitis in Matanzas, Cuba. In a number of cases no specific organism could be found, only broken down epithelial cells and mucous corpuscles; while in 70 per cent of the cases the pneumococcus was found, but not in great numbers. I can say that the observations of Dr. Gifford will apply to Matanzas, and that the pneumococcus of Fraenkel is the most frequent cause of conjuctivitis.

The number of cases of conjunctivitis is greatly increased in my regiment by the glaring sunlight and dust. The days are often intensely hot and the nights cool, in consequence, acute affections of all the mucous membranes are very common.

I mentioned the scarcity of cases of keratitis which came under observation. Relative to this I desire to note that five cases of keratitis presented themselves, in my regiment, following measles. Mumps and measles always follow a new army, especially so with regiments recruited from small towns and country districts. Men from the cities, when children, as a rule, pass through such diseases. While in private practice children, as a rule, are the ones that present eye-complications following measles. The soldiers could not be adequately protected from the light and would fail to take the necessary precautions which are enforced in private practice. There was nothing peculiar about the cases, except the slowness of their recovery, due to want of nutrition of the cornea. Such cases are irrigated with hot boracic solution, and antiseptic treatment of the ulcer.

The experience gained by long association with the army is invaluable to me, and my work kept me sufficiently in touch with our specialty. Fortunate, I think, is any surgeon who has, regardless of even great financial sacrifices, served with the American army during the Spanish-American war.

PERSISTENT PANNUS CURED AS A RESULT OF ACUTE DACRYOCYSTITIS.

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VERY many cases of trachoma associated with pannus seem, like Tennyson's brook, to go on forever, getting better, then worse, until the surgeon almost gives it up in despair. Through the kindness of Dr. Pollak the following case is presented, owing to the unusual complication which assisted materially in its cure.

Miss J. W., aged 12 years, came to the eye clinic of the Mullanphy Hospital, August 18, 1898. She is a delicate, anæmic girl who, according to the statement of her father, has had sore eyes from infancy. When about 7 years old she developed granular lids, and since that time (5 years) has received almost constant treatment, drifting from clinic to clinic.

When she came to the Mullanphy Hospital there was considerable lachrymation, photophobia and vision was reduced in the right eye to $^{20}/_{200}$ and in the left to $^{20}/_{100}$. On everting the lower lids the palpebral conjunctiva was seen to be thickened and roughened by partially absorbed trachoma granules. The conjunctiva was discolored a dark brown, the result of long-continued applications of nitrate of silver (argyrosis). The upper half of the cornea was overlaid with newly-formed vessels and tissue presenting a fleshy mass which hid the iris from view (pannus crassus). Atropine was instilled, and the inner surface of the lids touched with sulphate of copper three times a week. She was given cod-liver oil, and the following drops to use at home:

℞ Acid. tannic., - - - - - - gr. vijss.
Acid. borac., - - - - - - gr. xv.
Glycerin., - - - - - - ℥ij.
Aquæ, - - - - - - - ℥ij.

Having noticed a discharge from the nose, she was referred to Dr. Leo Chaplan, who found incipient atrophic rhinitis, and gave appropriate treatment. After several months of the above treatment, finding little or no benefit, my collegue, Dr. S. Pollak, suggested the use of jequirity. Eight beans were bruised and placed in one ounce of cold water, and allowed to stand 24 hours, then filtered. Two drops of this infusion was instilled into each eye three times a day for three days. This caused considerable reaction; pain, œdema of the lids and an abundant discharge of pus. The after-treatment consisted in the use of atropine and in frequent bathing with hot water.

After ten days the inflammation subsided, leaving the left cornea almost free from pannus, while the right cornea showed marked improvement. The improvement did not last, for after catching cold she returned almost as bad as ever. One morning our patient appeared with an acute dacryocystitis of the right side. There was severe pain, tense swelling of the sac and marked œdema and redness of the lids and surrounding parts. An incision was made into the sac, giving vent to a thick, creamy pus. After the inflammation had subsided the nasal duct was probed and syringed with an antiseptic solution. Our little patient did not take kindly to this treatment and avoided us for about two weeks, when the acute symptoms had recurred and it was necessary to evacuate the pus and continue the treatment as before.

This treatment, however, was carried out in a perfunctory manner, as it was observed that from the onset of the dacryocystitis a remarkable change for the better had taken place in the condition of the eye itself. The lachrymation and photophobia disappeared and the dense pannus began to clear up slowly but surely until, in a few weeks, had practically disappeared.

NOTE.—April 20, 1899: The patient has been seen at least twice a week since the above report was made. The cornea remains clear and the eyes look remarkably well. The only treatment consists in probing the nasal duct and syringing with an antiseptic solution.